

development of the disease in heterozygotes and homozygotes but these suggestions are unproven.

In light of our limited knowledge, all we can offer these patients at present is genetic counseling, advice about elimination of irritants, and the usual therapy of emphysema. However, studies on the biochemistry of α_1 -antitrypsin offer some hope that it may be possible to design a drug to replace the missing protein. With some luck such a drug may prevent the emphysema which develops in these patients.

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REFERENCES

- Kueppers F: α_1 -antitrypsin: Physiology, genetics, and pathology. *Humangenetik* 11:177-189, 1971
Black LF, Hyatt RE, Stubbs SE: Mechanism of expiratory airflow limitation in chronic obstructive pulmonary disease associated with α_1 -antitrypsin deficiency. *Am Rev Resp Dis* 105: 891-899, June 1972

Hypoxemic, Hyperventilatory Respiratory Insufficiency: A Common Post-operative Complication

WITH THE ADVENT of modern surgical techniques the morbidity and mortality associated with the intra-operative period has considerably declined. The post-operative period, however, is still fraught with many and diverse hazards, not the least of which is a fairly recently appreciated entity—hypoxemic, hyperventilatory respiratory failure. By definition this condition is associated with a PaO_2 below 60 mm of mercury, a PaCO_2 below 35 mm of mercury and a variable arterial pH. No patients or types of operation are immune. The etiology is multifactorial and may include one or a combination of the following: (1) Excessive intravenous fluid administration, (2) microscopic pulmonary emboli of clotted material or fat, (3) excessive supplemental oxygenation without ample humidification, (4) atelectasis, (5) anesthesia, (6) respiratory infections, (7) shock and (8) aspiration. These varied insults find expression at the pulmonary level as: mucociliary dysfunction; surfactant loss; macrophage disintegration; alveolar fluid filling and collapse; fluid and protein deposition in the alveolar capillary interstitium; and abnormal capillary blood flow and distribution. The physiologic expressions of these anatomical abnormalities are an abnormal distribution of both ventilation and perfusion, a decrease in lung compliance, an increase in airway resistance and a pronounced increase in the work of breathing—all manifested clinically as tachypnea, de-

creased tidal volume, hypoxemia, hypocapnia and a variable arterial pH.

Since this type of respiratory insufficiency has multiple causes, it would seem easier to prevent their occurrence than to correct them after the fact. This being so, *anticipatory* and *prophylactic* treatment would seem indicated. Thus, fluid and electrolytes should be carefully monitored particularly with an eye to preventing excess fluid loads; operative ventilatory support should not lead to under-ventilation or over-ventilation; anesthesia should be properly selected and carefully used; well humidified oxygenation should be appropriate to the patient's clinical situation; relief of pain should be accomplished with the least possible amount of analgesic, and pulmonary toilet and intermittent positive pressure breathing should be employed early and frequently. The patient's clinical condition should be followed at least daily by bedside tidal volumes and vital capacities, respiratory rates, physical examinations and arterial blood gases. If all these means are employed, hypoxemic hypocapnic ventilatory failure should decrease in frequency.

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REFERENCES

- Gump FE, Yoshiya M, Kinney JM: Water balance and extra-vascular lung water measurements in surgical patients. *Am J Surg* 119:515-518, 1970
Collins JA: The causes of progressive pulmonary insufficiency in surgical patients. *J Surg Res* 12:685-704, 1969

Early Diagnosis of Chronic Obstructive Pulmonary Disease

SINCE THE ADVANCED STAGE of the chronic obstructive pulmonary diseases (emphysema, chronic bronchitis, chronic asthma, rarely, bronchiectasis) appears to follow a relentless and progressive course regardless of treatment, increasing attention has been directed to early detection. Efforts are under way to screen large populations for α_1 -antitrypsin deficiency to detect high-risk individuals. Other workers are using newer screening pulmonary function tests to detect asymptomatic persons with early disease that still may be reversible. These results suggest that airway obstruction can be detected before it causes symptoms, and also that it is possible to differentiate one type of airway obstruction from another.

Even when the usual tests of pulmonary function are normal, airway obstruction can be detected by an abnormal decrease in maximal airflow rates, especially at low lung volumes. The association of a decreased single-breath CO diffus-

ing capacity suggests a loss of alveolar-capillary surface area (for example, emphysema). A normal diffusing capacity in the presence of airway obstruction suggests intrinsic airway disease. The specific type of airway disease may be defined by tantalum bronchography and pulmonary function tests in which airway resistance and maximal airflow rates are related to lung elastic recoil pressure. Finally, when all other pulmonary function tests are normal in an asymptomatic person, the "closing volume" may still be abnormally increased. This may indicate obstruction in the very smallest airways, less than 2 mm in diameter. If these different types of chronic obstructive pulmonary diseases can be detected early, preliminary studies suggest that therapeutic intervention may correct functional abnormalities; whether the disease itself is arrested or reversed remains to be seen.

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REFERENCES

- Gelf AFF, Gold WM, Wright RR, et al: Physiologic diagnosis of subclinical emphysema. *Am Rev Resp Dis* 107:50-63, 1973
- McCarthy DS, Spencer R, Green R, et al: Measurement of "closing volume" as a simple and sensitive test for early detection of small airway disease. *Am J Med* 52:747-753, Jun 1972
- McFadden ER, Jr, Linden DA: A reduction in maximum mid-expiratory flow rate—A spiographic manifestation of small airway disease. *Am J Med* 52:725-737, Jun 1972

Corticosteroids in the Treatment of Adult Respiratory Distress Syndromes

WHEN PATIENTS have experienced prolonged shock due to sepsis, hemorrhage, or following severe trauma, certain morphologic alterations in the lung are potential consequences. These may include intravascular platelet aggregation, sequestration of polymorphonuclear leukocytes with resultant release of lysosomal enzymes from these cells onto the pre-capillary arteriolar and capillary walls, and attenuation of pulmonary surfactant. These changes then lead to vascular constriction with leakage of plasma fluid into the pulmonary interstitial spaces. The end result of these well known changes is the development of functional alterations in pulmonary gas exchange characterized by progressively increasing hypoxemia and hypocapnea. Some of these same findings have also been described in pulmonary aspiration, smoke inhalation, and fat embolism.

The administration of high pharmacologic doses of corticosteroids has been suggested as desirable in preventing progression of the morphologic changes in the lung. They are thought to be particularly helpful in shock due to sepsis or hemorrhage, in smoke inhalations, or in aspiration pneu-

monia. Whether or not they are truly effective in adult respiratory distress syndromes of all types has not been conclusively established.

In order to be effective, the following dosage schedules have been recommended: Methylprednisolone 30 mg per kilogram of body weight or dexamethasone 6.0 mg per kilogram given either as a single bolus intravenous injection or repeated every eight hours for up to 72 hours.

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REFERENCES

- Christy JH: Treatment of gram-negative shock. *Am J Med* 50:77-88, Jan 1971
- Dietzman RH, Castaneda AR, Lillehei CW, et al: Corticosteroids as effective vasodilators in treatment of low output syndromes. *Chest* 57:440-453, 1970
- Wilson JW: Treatment or prevention of pulmonary cellular damage with pharmacologic doses of corticosteroid. *Surg Gynecol Obstet* 134:675-681, Apr 1972
- Blaisdell FW, Lim RC Jr, Stallone RJ: The mechanism of pulmonary damage following traumatic shock. *Surg Gynecol Obstet* 130:15-22, 1970
- McConn R, Del Guercio LRM: Respiratory function of blood in the acutely ill patient and the effect of steroids. *Ann Surg* 174:436-450, Sep 1971

Reversed Aorta to Saphenous Vein Grafts

WHEN THE CORONARY ARTERIES become occluded by atherosclerosis so that chest pain upon exertion, emotional reaction or after the ingestion of a large meal intervenes at regular intervals (stable angina) and does not respond to medical management, it is now an acceptable procedure to subject such patients to a series of studies including ventriculography and ciné coronary angiography. If they are found to be satisfactory candidates from all points of view, myocardial revascularization can be carried out by aorta-to-coronary vein grafting. Recent studies have indicated in-hospital mortalities of less than 7 percent throughout the country for single and double grafts and of 8 percent for triple grafts. Such experienced institutions as the Cleveland Clinic report a 1.2 percent mortality. Combinations with other revascularization procedures such as internal mammary-to-coronary anastomosis and internal mammary artery implantation have mortality rates similar to those of vein grafts alone, as does conventional coronary endarterectomies in certain selected cases.

Pre-infarction angina (Sampson's angina) is now also considered an indication for immediate myocardial revascularization. This differs from stable angina in that the chest pain lasts longer, has either not been present formerly or has changed in nature from its former pattern and is associated with transient changes in the T-wave